

The opinion in support of the decision being entered today was *not* written for publication and is *not* binding precedent of the Board.

**UNITED STATES PATENT AND TRADEMARK OFFICE**

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**BEFORE THE BOARD OF PATENT APPEALS  
AND INTERFERENCES**

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*Ex parte* KEVIN P. BAKER, DAVID BOTSTEIN,  
LUC DESNOYERS, DAN L. EATON, NAPOLEONE FERRARA,  
SHERMAN FONG, WEI-QIANG GAO, AUDREY GODDARD, PAUL J.  
GODOWSKI, J. CHRISTOPHER GRIMALDI, AUSTIN L. GURNEY,  
KENNETH J. HILLAN, JAMES PAN, NICHOLAS F. PAONI,  
MARGARET ANN ROY, VICTORIA SMITH, TIMOTHY A. STEWART,  
DANIEL TUMAS, COLIN K. WATANABE, P. MICKEY WILLIAMS,  
and WILLIAM I. WOOD

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Appeal 2007-1068  
Application 10/015,394  
Technology Center 1600

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DECIDED: May 14, 2007

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Before TONI R. SCHEINER, ERIC GRIMES, and NANCY J. LINCK,  
*Administrative Patent Judges*.

SCHEINER, *Administrative Patent Judge*.

**DECISION ON APPEAL**

This appeal under 35 U.S.C. § 134 involves claims to antibodies directed to a polypeptide designated PRO1760, which the Examiner has rejected for lack of patentable utility, and lack of enablement. We have jurisdiction under 35 U.S.C. § 6(b). We affirm.

## BACKGROUND

Initially, we note that this appeal is related to an appeal in Application No. 10/013,913 (Appeal No. 2006-1907). We have considered the two appeals together.

The Specification discloses a large number of cDNA clones (derived from various mammalian recombinant DNA libraries) encoding “PRO polypeptides” (Specification 6-280). The clone designated DNA76532-1702 (SEQ ID NO: 375) encodes PRO1760 (SEQ ID NO: 376), a polypeptide with “limited sequence identities to known proteins” (*id.* at 298: 23-27; 352: 17-20; Figs. 219 and 220). PRO1760 is one of eight PRO polypeptides that “tested positive as inhibitors of glucose and/or FFA uptake” in an assay in primary rat adipocytes (*id.* at 511: 34 to 512: 10). Eight other PRO polypeptides “tested positive as stimulators of glucose and/or FFA uptake” in the same assay (*id.*). According to the Specification, “PRO polypeptides testing positive in this assay would be expected to be useful for the therapeutic treatment of disorders where either the stimulation or inhibition of glucose uptake by adipocytes would be beneficial including, for example, obesity, diabetes or hyper- or hypo-insulinemia” (*id.*).

The Specification also teaches that antibodies to PRO polypeptides, in general, may be used as affinity purification reagents (*id.* at 380: 15-21); diagnostic reagents (*id.* at 380: 1-13); and antagonists of PRO polypeptides (*id.* at 371: 27-30).

## DISCUSSION

### THE CLAIMS

Claims 28-32 are pending and on appeal.

The claims stand or fall together, as Appellants have not made separate arguments for their patentability. 37 C.F.R. § 41.37(c)(1)(vii).

We will focus on claim 28 as representative:

28. An isolated antibody that specifically binds to the polypeptide of SEQ ID NO:376.

### UTILITY and ENABLEMENT

The Examiner rejected claims 28-32 as lacking utility sufficient to satisfy 35 U.S.C. § 101. The Examiner also rejected the claims as lacking enablement under 35 U.S.C. § 112, first paragraph. As the enablement rejection is largely a corollary of the Examiner's finding of lack of utility, our conclusion with respect to the § 101 issue will also apply to the § 112 issue.

Section 101 requires a utility that is both substantial and specific.

*In re Fisher*, 421 F.3d 1365, 1371, 76 USPQ2d 1225, 1229 (Fed. Cir. 2005). A substantial utility is one that “show[s] that an invention is useful to the public as disclosed in its current form, not that it may prove useful at some future date after further research. Simply put, to satisfy the ‘substantial’ utility requirement, an asserted use must show that that claimed invention has a significant and presently available benefit to the public.” *Id.*, 76 USPQ2d at 1230. A specific utility is “a use which is not so vague as to be meaningless.” *Id.* In other words, “in addition to providing a ‘substantial’ utility, an asserted use must also show that th[e] claimed invention can be

used to provide a well-defined and particular benefit to the public.” *Id.* The Examiner bears the initial burden of showing that a claimed invention lacks patentable utility. *See In re Brana*, 51 F.3d 1560, 1566, 34 USPQ2d 1436, 1441 (Fed. Cir. 1995).

The present claims are directed to antibodies specific for PRO1760. According to Appellants, the “utility of the claimed antibodies that bind PRO1760 is based upon the results of the adipocyte glucose/FFA uptake assay for the PRO1760 polypeptide” (Brief 4).

In the present case, no relationship between PRO1760 and any other known protein is disclosed. However, PRO1760 was one of several PRO polypeptides identified as a glucose or FFA uptake inhibitor in a glucose or FFA uptake assay in primary rat adipocytes (Specification 511: 34 to 512: 10). According to the Specification, any PRO polypeptide testing positive in the glucose or FFA uptake assay “would be expected to be useful for the therapeutic treatment of disorders where either the stimulation or inhibition of glucose uptake by adipocytes would be beneficial including, for example, obesity, diabetes or hyper- or hypo-insulinemia” (*id.*). Finally, the Specification teaches that antibodies to PRO polypeptides may be used as affinity purification reagents (*id.* at 380: 15-21); diagnostic reagents (*id.* at 380: 1-13); and antagonists of PRO polypeptides (*id.* at 371: 27-30).

The Examiner found that the mere identification of PRO1760 as an inhibitor of glucose- and/or free fatty acid-uptake is not sufficient to establish “a credible, specific and substantial asserted utility or a well established utility” for the polypeptide, or by extension, the antibody that specifically binds it (Answer 3). The Examiner acknowledges that

“PRO1760 scored positive as [an] *inhibitor* of glucose or FFA (free fatty acid) uptake in rat adipocyte cells” (*id.* at 4), but argues that the Specification does not explain how PRO1760 would be useful in treating diabetes, obesity, hyper- or hypo-insulinemia, as asserted in the Specification, “because in these conditions little or no glucose is entering the cells to begin with” (*id.*). Rather, the Examiner argues, “one skilled in the art would want to enhance glucose uptake” in these conditions (*id.* at 7).

Appellants argue that “[t]he fact that PRO1760 inhibits glucose uptake does not make it useless in such treatment. One of skill in the art would readily understand that *a protein which inhibits glucose uptake into adipocytes is a useful therapeutic target*, since blocking the function of this protein would decrease the inhibition, and thus increase glucose uptake into adipocytes” (Brief 4, emphasis in original). Appellants argue that “[o]ne of skill in the art would further understand that *antagonists to the PRO1760 polypeptide include antibodies*, such as the claimed antibodies which specifically bind the PRO1760 polypeptide” (*id.*).

This argument does not persuade us that the Examiner’s rejection should be reversed. To satisfy the requirements of § 101, a utility must be one that makes the invention useful to the public in its current form, not potentially useful in the future after further research. *See Fisher*, 421 F.3d at 1370, 76 USPQ2d at 1230. While it might be reasonable to infer that a given stimulator of glucose uptake would enhance glucose uptake in diabetes, obesity, hyper- and hypo-insulinemia, even if the stimulator had no specific role in any of those conditions, it does not follow that blocking the activity of a glucose uptake inhibitor would enhance glucose uptake - unless

the inhibitor is involved in the decreased uptake seen in those conditions in the first place. In the absence of any evidence that PRO1760 is involved in the decreased glucose uptake seen in diabetes, obesity, hyper- or hypo-insulinemia, the purely hypothetical possibility that blocking its inhibitory activity with antibodies might be useful in treating any of these conditions is too tenuous to establish “a significant and presently available benefit to the public” (*Fisher*, 421 F.3d at 1371, 76 USPQ2d at 1230).

Relying on Mueller I<sup>1</sup> and Mueller II<sup>2</sup> as evidence, Appellants also argue that “PRO1760, as an inhibitor of adipocyte glucose uptake, . . . has utility as a pharmacological tool for investigation of leptin regulation” (Brief 4), just like other inhibitors of glucose uptake “already known and used in the art such as 2-DG [(2-deoxy-D-glucose)], phloretin, and cytocholasin B” (*id.*), as well as stimulators like metformin and vanadium (*id.*).

We do not find this argument persuasive either. As both Mueller references make clear, all of these inhibitors and stimulators inhibited leptin secretion under assay conditions (Mueller I 557, col. 1; Mueller II 530, col. 2), and their usefulness as pharmacological tools in investigating leptin regulation depends upon an understanding of their mechanisms of action. For example, Mueller II suggests that “the effect of glucose utilization to stimulate leptin production involves the metabolism of glucose to a fate

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<sup>1</sup> W.M. Mueller et al., “Evidence That Glucose Metabolism Regulates Leptin Secretion from Cultured Rat Adipocytes,” *Endocrinology*, Vol. 139, No. 2, pp. 551-558 (1998), made of record January 10, 2005.

<sup>2</sup> W.M. Mueller et al., “Effects of Metformin and Vanadium on Leptin Secretion from Cultured Rat Adipocytes,” *Obesity Research*, Vol. 8, No. 7, pp. 530-539 (October 2000), made of record January 10, 2005.

other than anaerobic lactate production, possibly oxidation or lipogenesis” (Mueller II 530, col. 2), “[r]ather than glucose uptake per se” (*id.* at 531, col. 1), based on “the multiple known biological actions of vanadium” (*id.* at 537, col. 2).

Similarly, Mueller I teaches that “[t]he competitive inhibition [of leptin secretion] produced by 2-DG could be reversed by the addition of a high concentration of glucose, suggesting that 2-DG did not inhibit leptin secretion via a nonspecific toxic effect” (Mueller I 557, col. 1), while “the inhibition by phloretin was not reversed by glucose, as phloretin is not a competitive inhibitor” (*id.*).

Appellants have not explained how the observation that PRO1760 inhibits glucose uptake in adipocytes, with nothing more, is useful in investigating leptin regulation - especially as the evidence of record shows that leptin secretion can be inhibited by both inhibitors *and* stimulators of glucose uptake. That being the case, the mere identification of PRO1760 as a glucose uptake inhibitor does not provide a specific, well-defined, and particular benefit with respect to investigating leptin regulation.

We therefore conclude that neither the Specification’s disclosure, nor the extrinsic evidence relied on by Appellants, satisfies the utility requirement of 35 U.S.C. § 101 with respect to PRO1760, or antibodies that specifically bind it. The rejections of claims 28-35 and 38-40 under 35 U.S.C. §§ 101 and 112, first paragraph, are affirmed.

## SUMMARY

The Specification does not disclose a specific and substantial utility for the PRO1760-specific antibodies of claims 28-32, as required by 35 U.S.C. § 101. We therefore affirm the Examiner's rejection of the claims under § 101, and the corresponding rejection under § 112, first paragraph.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a)(1)(iv) (2006).

**AFFIRMED**

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